Childhood Lead Poisoning in Massachusetts Communities: Its Association with Sociodemographic and Housing Characteristics

ABSTRACT

Objectives. The purpose of the study was to examine the relationship between communities' sociodemographic and housing characteristics and incidence of lead poisoning.

Methods. This was a population-based correlational study of 238 275 Massachusetts children from birth through 4 years of age who were screened for lead poisoning in 1991–1992. A logistic regression model was developed with the community as the unit of analysis, the case identification rate for lead poisoning (newly identified children with venous blood lead ≥25 μg/dL per 1000 children) as the dependent variable, and US census variables as independent variables.

Results. A significant independent relationship with the community case identification rate of lead poisoning was found for seven variables: median per capita income, percentage of housing built before 1950, percentage of the population who were Black, percentage of children screened, and a "poverty index." Rates of iron deficiency and percentage of Hispanics were not associated with the case identification rate of lead poisoning.

Conclusions. Massachusetts communities' incidence of lead poisoning is correlated with sociodemographic and housing characteristics. In states similar to Massachusetts and without screening data, this model may help target screening programs. (*Am J Public Health.* 1995;85:528–534)

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Introduction

In October of 1991, the Centers for Disease Control (CDC) lowered the threshold for concern for blood lead elevation in children to 10 μg/dL, a level that defines as many as 10 million US children as at risk for lead toxicity. The CDC report stresses that only through communitywide interventions can blood lead levels in this range be prevented and suggests that prevention resources, including education and screening, should be targeted to those "communities of concern" where large numbers of children have blood lead levels of 10 μg/dL or higher.[1] In areas with screening programs for childhood lead poisoning, data identifying areas of high incidence are available; however, to our knowledge, no method of determining community risk without prior screening data has been described. State and local agencies in areas without prevention programs for childhood lead poisoning are therefore faced with the task of allocating resources without prior knowledge of incidence.

In March of 1990, Massachusetts passed a law (105 CMR 460.050) requiring yearly universal screening of children aged 9 months through 4 years for lead poisoning. The purpose of this paper is to describe the Massachusetts screening data at the state and community levels for children from birth through 4 years of age and to present the association at the community level between the case identification rate for lead poisoning and sociodemographic and housing variables from the 1990 census. We attempt to develop a model that will predict the community case identification rate for lead poisoning, using the census characteristics of a community.

Methods

Screening for lead poisoning was performed at a number of sites during the study period, including physicians' offices, hospitals, state-funded screening sites, and nutritional supplementation programs. In addition, the state sponsored several door-to-door screening projects in high-risk areas. Approximately 85% of all screening samples were analyzed at the Massachusetts or Boston Lead Laboratories, where analysis of capillary blood for erythrocyte protoporphyrin (EP) by hematofluorometry[2] was the primary screening test for lead poisoning. Blood lead analysis was performed for all capillary screening samples with levels of EP equal to or higher than 35 μg/dL. The state pursued venous confirmation samples in all children whose capillary samples showed blood lead levels of 25 μg/dL or higher. Blood samples were analyzed for lead by means of atomic absorption spectrophotometry.[3]

Definitions

The study period corresponded to the first year of mandatory screening in

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Massachusetts (April 1990 through March 1991). The population included all Massachusetts children from birth through 4 years of age who had an assay for EP or blood lead either analyzed by or reported to the Massachusetts Childhood Lead Poisoning Prevention Program during the study period. Screening rates were calculated by dividing the number of children screened by the total number of children from birth through 4 years of age in a given geographic area as determined by the 1990 US census. A venous blood lead concentration of 25 μg/dL or higher was considered indicative of lead poisoning. A case of lead poisoning was defined as any newly identified child from birth through 4 years of age with a venous blood lead concentration of 25 μg/dL or higher. Case identification rates for lead poisoning were calculated by dividing the number of cases by the number of children from birth through 4 years of age in a geographic area.

In young children, EP elevation is commonly associated with iron deficiency, lead poisoning, or both[4,5]; a blood sample with an EP concentration equal to or higher than 35 μg/dL *and* blood lead lower than 25 μg/dL was taken as an indicator of iron deficiency. For a given community, the rate of iron deficiency in the absence of lead poisoning was determined by dividing number of children with iron deficiency by the screened population.

Massachusetts Data

The following data were collected on all samples: test data, sample type (capillary or venous), EP result, and lead result if a blood lead analysis was performed. Name, address, and date of birth were recorded for all children at the time they were tested. To create a statewide data set, records from Boston, records from Children's Hospital in Boston, and EP screening data from the city of Worcester (performed at a state screening site there) were merged with records from the rest of the state. About 2% of screening samples were analyzed by private laboratories and the results reported to the Massachusetts Childhood Lead Poisoning Prevention Program for direct entry into the screening database. This created a master file with data on 303 982 children. Children with missing community data (n = 1115), those residing outside Massachusetts (n = 6692), those with age missing (n = 10 311), and those aged 5 years or older (n = 47 589) were excluded, resulting in a final analysis file that contained test analysis, age, and address data for 238 275 children. For children with multiple tests, results from the first (earliest date in the study period) sample were used in the analysis.

Children with lead poisoning were identified by searching the screening analysis file for children with a venous blood lead sample equal to or higher than 25 μg/dL. Additional cases of newly identified children were collected through four regional sites responsible for identifying and following all children with venous elevation in this range. Workers from these sites also identified for deletion from the file children with blood lead elevation prior to the study period. Finally, each record in this file was reviewed by one of the investigators (M.J.B.) who had first-hand knowledge of the population of poisoned children during the study period to confirm a final count of 862 children with newly identified lead poisoning.

Census Data

Population counts and sociodemographic characteristics of Massachusetts communities were obtained from the 1990 US Bureau of the Census STF 3A tape. Since deteriorating paint on pre-1950s housing represents the most important environmental source of lead in children,[1] we hypothesized that communities with high rates of poverty and old housing would have high rates of lead poisoning, while more affluent communities with low rates of old housing would have lower rates of lead poisoning.

Independent Variables

We examined the association between the community case identification rate for lead poisoning and independent variables describing demographics (percentage of the population who were Black, percentage who were Hispanic, population density), socioeconomic status (percentage of female-headed households with children younger than 18 years of age, percentage of high school dropouts, median per capita income, percentage of children younger than 5 years in poverty [i.e., living in households where the combined income was below the official poverty level for a family of that size], percentage of households with public assistance income), housing environment (percentage of homes not owner occupied, percentage of houses built before 1950), and physiologic status (rate of iron deficiency).

Communities were self-selected for screening, which means the children should not be considered a strictly random sample of the population. It follows that the case identification rates may be biased. In an attempt to minimize this effect, we included the within-community screening rate as a covariate in the bivariate analysis and in the logistic regression. Thus, we report a "screening adjusted" case identification rate.

Statistical Analysis

For bivariate associations of community characteristics and lead poisoning, the communities were aggregated into low, medium, and high groups with respect to each variable. In constructing these groups, communities were first sorted with respect to the independent variable; then cutoff values were determined that divided the population of children into three groups, each of which contained roughly one third of the total screened population. For each independent variable, we computed crude odds ratios and odds ratios adjusted for screening rate.

The association between each community characteristic and the community case identification rate for lead poisoning was also evaluated with a multiple logistic regression model in a stepwise fashion comparable to forward selection. Only those factors significant at the $\alpha = .05$ level were included in the final model, for which actual values of the community variables were used, as opposed to the grouped values used for the bivariate associations.

The Statistical Analysis System (SAS Institute Inc, Cary, NC) provides a logistic regression procedure, LOGIST, which was used to analyze the data in this study; the number of events was equal to the number of children with lead poisoning in each community and the number at risk was equal to the population count.

We assessed the fit of the logistic regression model with Somers' D statistic, an asymmetric measure of association for ordinal variables.[6] It is a modification of Kendall's tau-*b*. As such, it is similar to a correlation coefficient. It measures the correlation between the predicted values of the logistic regression and the 0 or 1 outcome. Perfect prediction yields $D = 1.0$. An alternative interpretation is in terms of a receiver operating characteristic (ROC) curve. A correct prediction of an outcome of 1 indicates a sensitive model and a correct prediction of an outcome of 0 indicates a specific model.

TABLE 1—Census Population, Number of Children Screened for Lead Poisoning, and Screening Rate, by Age, Massachusetts, April 1990 through March 1991

Age, y	1990 Population	No. of Children Screened	Screening Rate[a] (per 100)
<1	73 159	36 717	50.2
1–2	173 718	107 349	61.8
3–4	163 797	94 210	57.5
0–4	410 674	238 275	58.0

[a]Number of children screened divided by the total number of children (from the 1990 US census).

The complete set of predictions generates the ROC curve. The area under the ROC curve (AUC) is given by AUC = $(1 + D)/2$. Since the value of AUC > 0.5 is interpreted as the "information" in the data, D can be interpreted as the proportion of possible information explained by the logistic regression model.

Results

During the study period, 238 275 children from birth through 4 years of age were screened for lead poisoning in Massachusetts (Table 1), an overall screening rate of 58.0%. Massachusetts screening rates were highest in the 1- to 2-year age group (61.8%) and lowest in the younger-than-1-year age group (50.2%). Community screening rates varied from 3.7% to 97.2%; nevertheless, 284 of 350 Massachusetts communities fell within the same tercile (33%–67%).

Eight hundred sixty-two Massachusetts children from birth through 4 years of age were newly identified with venous blood lead levels equal to or higher than 25 μg/dL during the study period, a case identification rate of 2.1 per 1000 children. The case identification rate was highest in children aged 1 to 2 years (3.5 per 1000), with children in this age group accounting for 67.8% of the cases, and lowest in children younger than 1 year (0.6 per 1000). Case identification rates for Boston were about three times higher than state rates for all age categories.

The case identification rate for the majority of Massachusetts communities (n = 237) was zero. These communities were typically small (118 had fewer than 400 children in this age group) and rural; 215 of these 237 communities (91%) had a population density of 500 or fewer persons per square kilometer, and 76 (33%) had a population density of 50 or fewer persons per square kilometer (compare Cambridge, Mass, with 5748 persons per square kilometer). Because of the small numbers of children screened (in 87 communities fewer than 100 children were screened), case identification rates for these communities were highly variable when positive. For example, Provincetown, with the highest case identification rate (38 per 1000), had only 26 children screened and 1 child with a venous blood lead level equal to or higher than 25 μg/dL.

After adjustment for differences in screening rate, all of the independent variables showed statistically significant associations with childhood lead poisoning (Table 2). In other analyses not reported here, odds ratios were compiled after Boston was excluded, and the results were similar to those presented in Table 2.

Percentage of female-headed households with children younger than 18 years had a strong association with lead poisoning. Children in communities with large numbers of young children living in poverty (>20%) exhibited lead poisoning rates that were 8.9 (95% confidence interval [CI] = 6.6, 12.1) times those seen in communities with childhood poverty rates below 5%.

Of the variables describing housing environment, the variable that measured the rate of old housing showed a strong association with childhood lead poisoning. Those children living in communities where houses not occupied by owners constituted more than 60% of the housing stock were also significantly more likely to have lead poisoning than children in communities where the majority of houses were owner occupied (odds ratio = 6.7, 95% CI = 5.2, 8.5). Finally, the rate of iron deficiency, the only physiological marker we investigated, had a modest but statistically significant direct association with lead poisoning.

Of the 12 variables entered into the logistic model, 7 retained significant independent associations with the case identification rate for poisoning at the community level: (1) Percentage of female-headed households with children younger than 18 years; (2) percentage of the population who were Black; (3) median per capita income; (4) percentage of children aged 5 years or younger in poverty; (5) percentage of homes not owner occupied; (6) percentage of housing built before 1950; and (7) screening rate. The analysis indicated that variables 1, 4, and 5 were related. Their effect was best characterized in terms of a "poverty scale," which is the sum of the values of each variable divided by 3. The Somers' D test for this model (.47) indicates modest predictive ability. Estimated changes in risk of lead poisoning (Table 3) and analysis of variation and parameter estimates (Table 4) are given for this model for each significant community characteristic. Variables not found to have a significant relationship with lead poisoning in this model include percentage of the population who were Hispanic, population density, percentage of high school dropouts, percentage of the population receiving public assistance, and rate of iron deficiency.

As an example, we use the logistic model to assess the risk of a single child for lead poisoning, then estimate the number of lead-poisoned children for Worcester, Mass, given the city's census characteristics (Table 5). Worcester has 10% female-headed households; 58% of houses built before 1950; 60% of houses not owner occupied; median per capita income of $13 393; 28% of children aged 5 years or younger in poverty; 4.5% of the population Black; and 76% of the children screened. Step 1 is to determine the poverty scale for this city. Step 2 is to compute the products of each census coefficient and its corresponding parameter (from Table 4), and then sum their products. Step 3 is to exponentiate this sum: $\exp(-5.66) = .0035$. Step 4 is to compute the ratio: $.0035/(1 + .0035) = .0035$. A child in Worcester therefore has a predicted probability of being lead poisoned of .0035, or 3.5 per 1000 children. To estimate the number of lead-poisoned children in Worcester predicted by this model, the probability for an individual child is multiplied by the census population, which in this case is 12 271 children from birth through 4 years of age: $.0035 \cdot 12\,271 = 43$.

To determine whether any geographic areas had rates of lead poisoning that were significantly higher or lower than the model predicted, we mapped standardized residuals for all Massachusetts communities. A visual examination of the mapped standardized residuals showed that communities in which lead poisoning was over- or underpredicted were evenly distributed across the state.

TABLE 2—Screening Rates, Case Identification Rates, and Odds Ratios for Lead Poisoning in Massachusetts Children from Birth through 4 Years of Age, by Community Characteristics, April 1990 through March 1991

	No. of Children[b]	No. of Children Screened	Screening Rate per 100 Children	Lead Poisoning[a] No.	Case Identification Rate[c] per 1000 Children	Crude OR (95% CI)	Adjusted OR[d] (95% CI)
% population Black							
<1	152 227	80 939	53	96	1.2	1.0	1.0
1–4	146 335	83 666	57	212	2.5	2.3 (1.8, 2.9)	2.1 (1.7, 2.7)
>4[e]	112 112	73 670	66	554	7.5	7.9 (6.3, 9.8)	6.0 (4.7, 7.5)
% population Hispanic							
<1	121 941	66 660	55	72	1.1	1.0	1.0
1–4	136 954	74 073	54	111	1.5	1.4 (1.0, 1.8)	1.4 (1.0, 1.9)
>4[e]	151 779	97 542	64	679	7.0	7.6 (6.0, 9.7)	6.2 (4.9, 8.0)
Population density[f]							
<500	159 017	83 282	52	97	1.2	1.0	1.0
500–1500	102 276	56 684	55	113	2.0	1.8 (1.4, 2.4)	1.7 (1.3, 2.3)
>1500[e]	149 381	98 309	66	652	6.6	7.2 (5.8, 8.9)	5.5 (4.8, 7.0)
% female-headed households with children < 18 y							
<5	133 609	70 660	53	65	0.9	1.0	1.0
5–10	169 794	94 886	56	220	2.3	2.7 (2.0, 3.5)	2.6 (1.9, 3.4)
>10[e]	107 271	72 729	68	577	7.9	11.1 (8.6, 14.4)	9.3 (7.0, 12.1)
% high school dropouts							
<10	99 969	52 808	53	45	0.9	1.0	1.0
10–20	224 196	134 578	60	521	3.9	5.2 (3.8, 7.0)	3.7 (2.7, 5.0)
>20[e]	86 509	50 889	59	296	5.8	7.6 (5.6, 10.4)	5.7 (4.1, 7.8)
Median per capita income, $							
>20 000	74 527	38 970	52	35	0.9	1.0	1.0
15 000–20 000	198 467	116 728	59	412	3.5	1.4 (1.3, 1.7)	1.3 (1.2, 1.5)
<15 000	137 680	82 577	60	415	5.0	6.3 (4.5, 9.1)	4.2 (2.9, 5.9)
% children ≤5 y in poverty							
<5	116 393	62 784	54	47	0.7	1.0	1.0
5–20	159 359	88 046	55	191	2.2	3.0 (2.2, 4.1)	2.9 (2.1, 3.9)
>20[e]	134 922	87 445	65	624	7.1	11.5 (8.6, 15.5)	8.9 (6.6, 12.1)
% population receiving public assistance							
<5	140 430	75 067	53	68	0.9	1.0	1.0
5–10	138 017	76 945	56	163	2.1	2.4 (1.8, 3.2)	2.3 (1.7, 3.1)
>10[e]	132 227	86 263	65	631	7.3	9.9 (7.7, 12.7)	8.0 (6.0, 10.1)
% houses not owner occupied							
<40	171 701	92 811	54	86	0.9	1.0	1.0
40–60	145 432	84 403	58	342	4.1	4.7 (1.3, 6.0)	4.0 (3.2, 5.2)
>60[e]	93 541	61 061	65	434	7.1	9.3 (7.4, 11.7)	6.7 (5.2, 8.5)
% housing built before 1950							
<40	159 538	85 001	53	68	0.8	1.0	1.0
40–60	143 950	82 886	58	342	4.1	5.6 (4.3, 7.2)	4.8 (3.7, 6.3)
>60[e]	107 186	70 388	66	452	6.4	9.9 (7.7, 12.8)	6.9 (5.3, 9.0)
Rate of iron deficiency (per 100 population)							
<4	137 791	80 064	58	122	1.5	1.0	1.0
4–6	169 548	96 737	57	412	4.3	2.7 (2.2, 3.4)	2.8 (2.3, 3.4)
>6[e]	103 335	61 474	59	328	5.3	3.6 (2.9, 4.4)	3.2 (2.6, 4.0)

Note. OR = odds ratio; CI = confidence interval.
[a]A case of lead poisoning was defined as a newly identified child with a venous blood lead level equal to or higher than 25 μg/dL.
[b]1990 US census figures.
[c]Number of cases divided by the number of children.
[d]Adjusted for the screening rate.
[e]This group included Boston.
[f]Number of people per square kilometer.

Discussion

This study is the first to examine childhood lead poisoning data in a state where children were screened in diverse urban, suburban, and rural communities. We found lead poisoning in all types of communities. Although lead poisoning is widespread in Massachusetts, those children living in communities with high rates of poverty, single-parent families, and pre-1950s housing and low rates of home ownership were 7 to 10 times more likely to have lead poisoning. These data support screening recommendations that tar-

TABLE 3—Independent Relationship between Significant Variables in the Logistic Model and Odds Ratio for Lead Poisoning (Blood Lead ≥ 25 μg/dL) at the Community Level

	Unit of Measurement	Independent Effect: Change in Odds Ratio	95% Confidence Interval
Median per capita income	$1000	0.92	0.89, 0.95
% population Black	1%	1.04	1.03, 1.05
Poverty scale[a]	1%	1.02	1.01, 1.04
% housing built before 1950	1%	1.02	1.01, 1.03
Screening rate	1%	1.02	1.01, 1.02

[a]The sum of the values for percentage of female-headed households with children younger than 18 years, percentage of children aged 5 years or younger in poverty, and percentage of houses not owner occupied divided by 3.

TABLE 4—Final Logistic Regression Model: Analysis of Variation

	Estimate	SE	*P*
Median per capita income	−0.0824	0.0175	.0001
% population Black	0.0364	0.0047	.0001
Poverty scale[a]	0.0255	0.0075	.0007
% housing built before 1950	0.0192	0.0038	.0001
Screening rate	0.0156	0.0036	.0001
Intercept	−7.86	0.4227	.0001

[a]The sum of the values for percentage of female-headed households with children younger than 18 years, percentage of children aged 5 years or younger in poverty, and percentage of houses not owner occupied divided by 3.

TABLE 5—Estimation of Risk for Lead Poisoning for an Individual Child Living in Worcester, Mass, Given the City's Census Characteristics

Parameter	Coefficient	Product
−0.0824	Median per capita income = $13 400	−1.10
0.0364	% Black = 4.5	0.16
0.0255	Poverty scale[a] = 33	0.84
0.0192	% pre-1950s housing = 58	1.11
0.0156	% screened = 76	1.19
−7.86	Baseline = 1.0	−7.86
Sum		−5.66

[a]The sum of the values for percentage of female-headed households with children younger than 18 years, percentage of children aged 5 years or younger in poverty, and percentage of houses not owner occupied divided by 3.

get poor children, such as the directive recently announced by the federal Health Care Financing Administration requiring lead screening for all Medicaid recipients between 6 and 72 months of age. Since the socioeconomic characteristics often required for Medicaid eligibility are the same that we found to be associated with lead poisoning, we should expect the population served by this program to have a high rate of lead poisoning.

Unfortunately, communities at highest risk for lead poisoning are often medically underserved areas. Since physician-driven screening misses large numbers of children at risk, it is important to use additional screening tactics, such as door-to-door screening by community nurses, in these communities. Our data may help states without prior experience with blood lead screening in children to target screening resources in high-risk communities. In Massachusetts, the use of just one census variable can identify communities where the likelihood of identifying children with lead poisoning is significantly increased; for example, there were 15 communities with more than 25% of children aged 5 years or younger living in poverty. These communities contained 34% (80 475) of the screened children and 71% (615) of all lead-poisoned children identified during the study period.

In accord with commonly held views, the logistic analysis points to an independent relationship between childhood lead poisoning and descriptors of old housing, poverty, and race. Lead from pre-1950s house paint is thought to be the most important source of lead poisoning in US children.[1] Supporting this view are numerous studies of individual children that confirm the importance of the ingestion of lead paint–contaminated house dust as the most important vehicle for lead ingestion.[7–9] Old houses with deteriorating lead paint from interior surfaces often have high dust lead contamination.[10–12] This analysis confirms the relationship of leaded paint and lead poisoning in a large population of children; percentage of houses built before 1950 displayed a robust, statistically significant direct relationship with the case identification rate for lead poisoning in all models we performed. The other housing variable, percentage of homes not owner occupied, also displayed a significant positive association with lead poisoning. On the basis of this association, we suggest that old homes that are not owner occupied are more likely to be allowed to deteriorate and to release lead-based paint into the environment. Taken in combination, these associations suggest that legislative efforts requiring abatement of lead-based paint directed toward pre-1950s houses that are not owner occupied would confer the greatest benefit to children in terms of lead poisoning prevention.

Communities with a higher proportion of Black children were at greater risk for childhood lead poisoning, confirming the results of other studies[13,14] where Black children were found to be at increased risk. Interestingly, the relative rate of lead poisoning for Blacks vs Whites for the second National Health and Nutrition Examination Survey (NHANES II) sample of children (6.4) was similar in magnitude to the adjusted odds ratio obtained in our study. There have been few epidemiological studies of lead poisoning in Hispanic children. Our data suggest that Hispanic children have a risk for lead poisoning (adjusted odds 6.2) that is similar to that seen in Black children. After the effects of poverty and housing status are controlled for, however, the relationship between percentage of the population who were Hispanic and lead poisoning loses its significance, while percentage of the population who were Black retains a statistically significant direct relationship with lead poisoning. We speculate that the increased incidence of lead poisoning in Hispanics, and much of the increased incidence in Blacks, is explained by the greater incidence of poverty and poor housing for these racial groups. This hypothesis is supported by findings in Boston that over many years the same buildings have housed successive populations of lead-poisoned children of Irish, Black, and now Hispanic descent.

In Blacks, however, additional biological variables may influence risk for lead poisoning. It has been known for some time that dietary calcium blocks the absorption of lead from the gastrointestinal tract.[15–17] An examination of NHANES II data showed that Black children had significantly lower calcium intakes than their White counterparts.[18] This may be due to the high rate of lactose intolerance in the Black population. Low calcium intake after the first year of life may, therefore, be an additional biological risk factor for lead poisoning, and may be responsible for some of the association between the percentage of the population who are Black and incidence of lead poisoning after the effects of poverty and poor housing are controlled for.

Finally, this logistic model may be employed in states without screening programs to target high-risk communities. While we recognize that the nonrandom selection of children for screening may bias our model of case identification rates, screening appears to have a direct and independent effect on the case identification rate. From this association we infer that the ordering of the communities with respect to risk is not affected by changes in the screening rate; that is, communities that are otherwise at high risk will remain at high risk when the screening rate rises. Hence, for states with no screening data, predicted values for communities may be generated from the logistic model, using our minimum screening rate (4%) as a constant rate across communities. Communities may then be ranked according to their respective predicted values. These values will reflect the underlying order of the communities in terms of their risk of childhood lead poisoning.

This study is subject to a number of limitations. We examined children with markedly elevated blood lead by current standards. Using the current threshold of concern (10 μg/dL), the number of children in Massachusetts with lead exposure is likely to be four to eight times the number of children identified in this study.[19] We cannot determine from these data whether the community characteristics of old housing and poverty are also linked with blood lead elevation in the 10 to 24 μg/dL range. Additionally, two factors result in an underestimate of the incidence of blood lead elevation in the range we studied. First, screening for lead poisoning with EP misses approximately half of children with blood lead equal to or higher than 25 μg/dL.[20] Second, we used census counts for rate denominator when, on average, only about 60% of children in any community were screened. It is also important to note that the regression model has not been validated in a separate population sample. Before this model is used in other locations, validation studies should be conducted. Finally, this model was developed to identify lead poisoning due to lead paint contamination; we would not expect its use to identify point sources of industrial contamination. However, a residual analysis similar to the one we performed might identify areas where industrial contamination is a factor in childhood lead poisoning.

Childhood lead poisoning is a preventable disease. The use of blood lead levels to screen children is a relatively inexpensive means of early detection of environmental lead exposure in children. This analysis indicates that community census variables describing socioeconomic and housing status correlate with the community risk of childhood lead poisoning. The regression model presented in this study may be useful in targeting screening efforts in high-risk communities for states similar to Massachusetts but without community-level data on lead poisoning incidence. □

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